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PNEUMOPERITONEUM FOLLOWING MECHANICAL VENTILATION IN A CHILD

Fazal H. Khan and M. Nadeem Ahmad¹

ABSTRACT

A case of spontaneous pneumoperitoneum in a 5 months old child is being described. This child developed massive pneumoperitoneum while being mechanically ventilated. This possibility should always be kept in mind specially if child develops pneumoperitoneum in the presence of very high airway pressures.

KEY WORDS: Ventilation. Pneumoperitoneum. Barotrauma.

INTRODUCTION

Tension pneumothorax and pneumomediastinum are well-recognised complications of pulmonary barotrauma. Pneumoperitoneum secondary to barotrauma is much less common.¹⁻⁴ Most cases of pneumoperitoneum associated with barotrauma are reported in adults, neonates and are not reported in pediatric age group.^{5,6} Prompt recognition and differentiation between surgical and non-surgical pneumoperitoneum can save a critically-ill child from undergoing unnecessary surgical exploration.

CASE REPORT

A 5 months old baby girl with a history of loose motions and fever for one week was presented in the emergency room of our hospital. Her past medical history was unremarkable. On examination she was restless, her heart rate was 140/min, respiratory rate was 65/min and oxygen saturation 59%. Her blood pressure was 100/60. She had a cardiopulmonary arrest in the emergency room. She was resuscitated and shifted to ICU and put on mechanical ventilation. Further examination and investigations revealed that she was suffering from rickets, pneumonia and hypocalcemia.

ICU COURSE

On the 1st day of admission, she was put on pressure control mode of ventilation. Her pressure control was set at 8 cms H₂O, respiratory rate was 20/min and F_IO₂ was kept at 0.5. Her arterial blood gases were showing a pH of 7.43, PaCO₂ of 32 mmHg, PaO₂ of 80 mmHg, HCO₃ was 21 meq/l and SaO₂ was 98%. Her chest x-ray revealed evidence of surgical emphysema in the neck and hyperinflation in both lungs, (Figure 1).

On the 3rd day in the I.C.U. deterioration in lung function was noted. Her chest x-ray revealed pneumomediastinum and irregular densities in the lung field suggesting onset of respiratory distress syndrome, (Figure 2).

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Figure 1: Chest x-ray showing hyperinflated lung fields and surgical emphysema in the neck

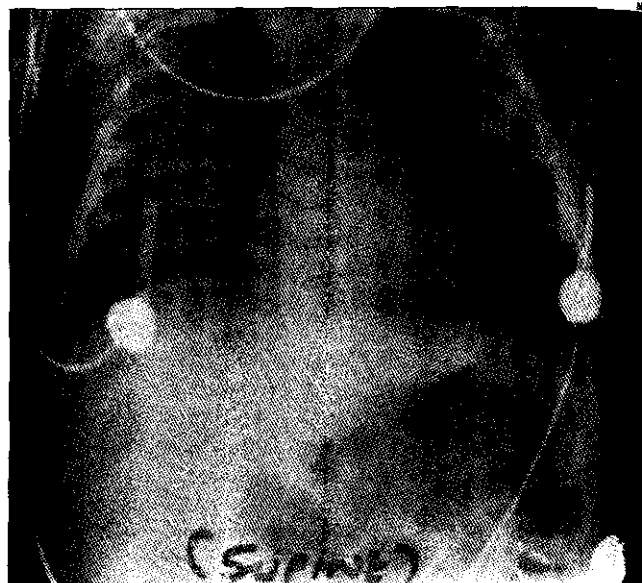


Figure 2: Chest x-ray suggesting onset of acute respiratory distress syndrome

Her arterial blood gases showed a pH of 7.25, PaCO_2 of 74 mmHg, PaO_2 of 51 mmHg and SaO_2 of 80%. Her ventilatory parameters were adjusted and she was put on a pressure control of 20 cm of H_2O , PEEP was gradually increased to 6 cm of H_2O and her F_1O_2 was increased subsequently to 100%. Her peak inspiratory pressure at that time was 28 cms of H_2O . On these parameters the repeat ABG's showed a pH of 7.47, PaCO_2 of 33 mmHg, PaO_2 of 80 mmHg, and SaO_2 was 96%. Her respiratory functions continued to deteriorate. During the next 4 days i.e., from day 4-7 following ICU admission, F_1O_2 and pressure control requirements remained high. She remained unstable requiring frequent adjustments in F_1O_2 and pressure control to keep PaO_2 around 60 mmHg and PaCO_2 in the range of 34-45 mmHg. On the 8th day of ICU admission the lung functions were so much deranged that she was requiring a pressure control of 30 cms of H_2O , PEEP of 12 cms of H_2O and F_1O_2 of 0.85. Even on these ventilatory parameters her pH was 7.36, PaCO_2 44 mmHg and SaO_2 was 90%. At that time peak inspiratory pressure was 40-42 cm of H_2O . Abdominal distension was noted in the evening and x-ray chest and abdomen revealed pulmonary interstitial emphysema and massive pneumoperitoneum (Figure 3). There was increase in the abdominal girth, the patient became hypotensive and was put on dopamine and dobutamine which restored the blood pressure. For the massive pneumoperitoneum surgical consultation was taken and exploratory laparotomy was planned. This decision was based on the evidence that patient was having elevated white cell count and coagulopathy. Pneumoperitoneum secondary to air leak from the thorax was also considered in the differential diagnosis. The patient's father did not agree for the operation so patient was managed conservatively. Abdominal girth gradually decreased. Patient was given surfactant. Improvement in lung compliance and decrease in the ventilatory requirements were observed. Peak

inspiratory pressure was decreased significantly along with the decrease in the requirement of positive end expiratory pressure. The ABGs were satisfactorily maintained on F_1O_2 of 0.5% and PEEP of 4 cm of water. Pneumoperitoneum was completely resolved eight days later. The patient was gradually weaned off from the ventilator, shifted out of ICU and then finally discharged for home. The patient is coming for follow-up regularly without any residual problems.

DISCUSSION

The main causes of accidental pneumoperitoneum are intestinal perforation, which accounts for 90% of cases and pulmonary barotrauma.⁷ In patients undergoing IPPV, pneumoperitoneum as a result of barotrauma has been reported in intensive care patients with ARDS.⁸ Pneumoperitoneum can also occur as a complication of CPR as well as transtracheal jet ventilation.^{9,10}

Potential extension of air from the mediastinum to abdomen is a well recognized physiological phenomenon. Experimental studies have shown that insufflation of air in the mediastinum of dogs leads to dissection of air to the retroperitoneum through the esophageal or aortic diaphragmatic hiatus along the paraaortic areolar tissue.¹¹ Weakness or congenital defects of the parietal peritoneum will sometimes allow the retroperitoneum air to enter the peritoneal cavity and produce pneumoperitoneum. Clinical and conventional radiographic observation have documented the occurrence of this phenomena in patients with spontaneous pneumomediastinum or pneumomediastinum associated with trauma or mechanically assisted ventilation.¹²⁻¹⁴

Alternatively the pleural space may communicate with the peritoneum through pleuro-peritoneal defect either congenitally or acquired, so tension pneumothorax may be associated with a tension pneumoperitoneum.

Under normal conditions the intra abdominal pressure exceeds the intrathoracic pressure by an average of 20-30 cm H_2O during both inspiration and expiration. Therefore, simple pneumothorax with or without subcutaneous emphysema should not cause a pneumoperitoneum.¹⁶

Mechanical ventilation as well as positive end expiratory pressure increase the intra thoracic pressure and consequently the risk of air dissection into the peritoneal cavity. It is recommended that if a child is requiring PEEP to improve oxygenation then PEEP should be gradually increased starting from 3 cms of H_2O . Changes in PEEP level should be taken in steps of 1-2 cms H_2O and in extreme cases PEEP of upto 10 cms H_2O can be applied. Higher values of PEEP are not tolerated well by children and results in hyperinflation. Our patient was on a PEEP of 12 cm H_2O and her peak inspiratory pressures were 40-42 cm of H_2O which could well explain the occurrence of pneumoperitoneum.

It is important to distinguish surgical from non-surgical pneumoperitoneum. Pneumoperitoneum secondary to pneumothorax is strongly suspected when the chest roentgenogram shows hyaline membrane disease with pulmonary interstitial emphysema, pneumothorax or pneumomediastinum.⁶ However, patients with pneumoperitoneum arising from the chest may have only mildly abnormal



Figure 3: X-ray showing massive pneumoperitoneum

chest roentgenograms.¹⁸ Vice versa, the patients with pulmonary interstitial emphysema, pneumothorax, and pneumomediastinum are ill and may have co-existing bowel perforation.

Several studies may aid in determining the cause of pneumoperitoneum. Analysis of the oxygen content of peritoneal gas may help. Chang has demonstrated > 60% oxygen in the peritoneal air of two ventilated infants receiving high concentration of oxygen.¹⁹ Problems with this approach are that the infant must be ventilated with a high gas mixture and one must assume that oxygen is not escaping around the ETT and being swallowed.

Water soluble iodinated radiographic contrast agents can be used in evaluation of bowel perforation.²⁰ The absence of a gastric air fluid level is a useful sign as it is found in > 90% of neonates with gastric perforation.^{21,22}

Usually the clinical status, laboratory findings and rapidity of abdominal distension will differentiate surgical from non-surgical pneumoperitoneum. Unfortunately, concomitant DIC and sepsis depresses the platelet count and the white blood count, further confusing the issue.

CONCLUSION

In conclusion it is recommended that a sequential approach to ventilated child with a pneumoperitoneum is to look for the presence of an intraperitoneal air fluid level on a true horizontal beam roentgenogram if present laparotomy is indicated. If a child has both, a posterior pneumomediastinum and a large pneumoperitoneum or a PIP of at least 30 cms of water with evidence of extra alveolar air and a large pneumoperitoneum, the child can be safely observed.

If a ventilated child with a pneumoperitoneum has no extra alveolar air dissection and a PIP of < 28 cms of water, laparotomy should be undertaken.²²

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